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NEWS	6	FEB 16	New FASTA Display Formats Added to USGENE and PCTGEN
NEWS	7	FEB 16	INPADOCDB and INPAFAMDB Enriched with New Content and Features
NEWS	8	FEB 16	INSPEC Adding Its Own IPC codes and Author's E-mail Addresses
NEWS	9	APR 02	CAS Registry Number Crossover Limits Increased to 500,000 in Key STN Databases
NEWS	10	APR 02	PATDPAFULL: Application and priority number formats enhanced
NEWS	11	APR 02	DWPI: New display format ALLSTR available
NEWS	12	APR 02	New Thesaurus Added to Derwent Databases for Smooth Sailing through U.S. Patent Codes
NEWS	13	APR 02	EMBASE Adds Unique Records from MEDLINE, Expanding Coverage back to 1948
NEWS	14	APR 07	CA/CAplus CLASS Display Streamlined with Removal of Pre-IPC 8 Data Fields
NEWS	15	APR 07	50,000 World Traditional Medicine (WTM) Patents Now Available in CAplus
NEWS	16	APR 07	MEDLINE Coverage Is Extended Back to 1947

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FILE 'HOME' ENTERED AT 12:06:56 ON 20 APR 2010

=> FIL BIOSIS CAPLUS EMBASE
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FULL ESTIMATED COST

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=> s (BLC or ELC) (3a) promoter
L1 3 (BLC OR ELC) (3A) PROMOTER

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 3 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L2 ANSWER 1 OF 3 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights
reserved on STN
AN 0019807029 EMBASE
CP MEDLINE® is the source for the citation and abstract of this record.
TI New putative control elements in the promoter OF CXCL13 chemokine gene, a
target of alternative NF-kappaB pathway.
AU Britanova, L.V. (correspondence); Kuprash, D.V.
SO Molekuliarnaia biologiia, (2009 Jul-Aug) Vol. 43, No. 4, pp. 657-665.

ISSN: 0026-8984
CY Russian Federation
DT Journal; Article
FS MEDLINE
LA Russian
ED Entered STN: 13 Apr 2010
Last Updated on STN: 13 Apr 2010
AB We searched the proximal promoter region of CXCL13/BLC chemokine gene for new putative control elements, including potential NF-kappaB binding sites. Using electrophoretic mobility shift assay and reporter gene analysis we identified two new promoter elements. The first element contains Rel/NF-kappaB binding site and seems to participate in inducible gene expression, while another site binds transcription factor Sp1 and is critical for basic transcription. It is the first indication that alternative NF-kappaB pathway target genes are probably cooperatively controlled by factors Rel/NF-kappaB and Sp1. Identification of a functional Sp1 site in the promoter of a target gene of alternative NF-kappaB pathway will be useful for investigation of molecular mechanisms and signal pathwaysinvolved.

L2 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:717163 CAPLUS
DN 137:380824
TI Dynamic changes in histone H3 Lys 9 methylation occurring at tightly regulated inducible inflammatory genes
AU Saccani, Simona; Natoli, Gioacchino
CS Institute for Research in Biomedicine, Bellinzona, CH6501, Switz.
SO Genes & Development (2002), 16(17), 2219-2224
CODEN: GEDEEP; ISSN: 0890-9369
PB Cold Spring Harbor Laboratory Press
DT Journal
LA English
AB Methylation of histone H3 at Lys 9 is causally linked to formation of heterochromatin and to long-term transcriptional repression. We report an unexpected pattern of H3 Lys 9 methylation occurring at a subset of inducible inflammatory genes. This pattern is characterized by relatively low constitutive levels of H3 Lys 9 methylation that are erased upon

activation and restored concurrently with post-induction transcriptional repression. Changes in H3 Lys 9 methylation strongly correlate with RNA polymerase II recruitment and release. In particular, remethylation correlates with RNApolII release more strongly than does histone deacetylation. We propose that, by generating a window of time in which transcription is permitted, dynamic modulation of H3 Lys 9 methylation adds an addnl. regulatory level to transcriptional activation of tightly controlled inducible genes.
OSC.G 84 THERE ARE 84 CAPLUS RECORDS THAT CITE THIS RECORD (84 CITINGS)
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1991:443480 CAPLUS
DN 115:43480
OREF 115:7437a,7440a
TI Synthetic genes for streptokinase and streptokinase analogs and their expression in Escherichia coli
IN Fujii, Setsuro; Katano, Tamiki; Majima, Eiji; Ogino, Koichi; Ono, Kenji;
Sakata, Yasuyo; Uenoyama, Tsutomu
PA Otsuka Pharmaceutical Factory, Inc., Japan
SO Eur. Pat. Appl., 76 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 407942	A2	19910116	EP 1990-113099	19900709
	EP 407942	A3	19910904		
	EP 407942	B1	19951011		
	R: AT, CH, DE, DK, ES, FR, GB, IT, LI, NL, SE				
	JP 04011892	A	19920116	JP 1990-179851	19900706
	US 5240845	A	19930831	US 1990-549049	19900706
	AU 9058806	A	19910117	AU 1990-58806	19900709
	AU 648029	B2	19940414		
	AT 129014	T	19951015	AT 1990-113099	19900709

```
     ES 2078925           T3    19960101    ES 1990-113099
19900709
     CA 2020828           A1    19910112    CA 1990-2020828
19900710
PRAI JP 1989-179432       A     19890711
     JP 1989-307957       A     19891127
     JP 1990-96830        A     19900411
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB   Genes encoding streptokinase (I) and its derivs. are synthesized
and
     expressed in a host such as Escherichia coli for manufacture of
I suitable for
     clin. application.  The DNA encoding natural-type I was
synthesized by
     standard chemical and used for construction of expression
plasmid pSKXT, which in
     turn expressed the I gene using the E. coli tac promoter and the
     blc signal sequence.  Efficient expression of the gene in the E.
     coli transformants and purification of the protein product were
demonstrated.
     I analogs with a carboxy-terminal deletions, optionally with
internal
     modifications were also described.
OSC.G   3     THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3
CITINGS)


=> FIL STNGUIDE
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      30.60      33.90

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                      -1.70
-1.70

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=> FIL BIOSIS CAPLUS EMBASE
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                       0.07      33.97

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00
-1.70
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=> s NF kapp B
L3            1 NF KAPP B

=> s NF kappa B
L4       113335 NF KAPPA B

=> s l4 and (Blc or Elc)
L5           36 L4 AND (BLC OR ELC)

=> s l5 and promoter
L6            5 L5 AND PROMOTER

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7             4 DUP REM L6 (1 DUPLICATE REMOVED)

=> d bib abs 1-y
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DISPLAY ACC is not allowed in a multifile environment.  Enter
"DISPLAY HISTORY" to locate the file the L# was created in, use the
FILE command to enter that file, and re-enter the DISPLAY ACC
command.

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L7   ANSWER 1 OF 4  BIOSIS  COPYRIGHT (c) 2010 The Thomson
Corporation  on STN
     DUPLICATE 1
AN   2009:551971  BIOSIS
DN   PREV200900553074
TI   New putative control elements in the promoter of the gene for
     the CXCL13 chemokine, a target of the alternative NF-
     kappa B pathway.
AU   Britanova, L. V. [Reprint Author]; Kuprash, D. V.
CS   Russian Acad Sci, VA Engelhardt Mol Biol Inst, Moscow 119991,
Russia
     kuprash@eimb.ru
SO   Molecular Biology (Moscow), (AUG 2009) Vol. 43, No. 4, pp.
604-611.
```

CODEN: MOLBBJ. ISSN: 0026-8933. E-ISSN: 1608-3245.
DT Article
LA English
ED Entered STN: 30 Sep 2009
Last Updated on STN: 30 Sep 2009
AB The proximal promoter region of the gene for the CXCL13/
BLC chemokine has been studied by electrophoretic mobility shift
assay and reporter gene analysis in order to detect new control
elements,
in particular, NF-kappa B binding sites.
Two new putative control elements have been identified. One of
them
contains a Rel/NF-kappa B binding site and
seems to participate in inducible gene expression. The other is
an Sp1
factor binding site, essential for basal transcription. It is
the first
time that such a site is found in the promoter of a target gene
of the alternative NF-kappa B pathway. This
finding indicates that genes under the control of the alternative
NF-kappa B pathway can be cooperatively
regulated by Rel/NF-kappa B and Sp1. Our
results will add to the understanding of the signaling pathways
that
govern the expression of genes controlled by the alternative NF-
kappa B pathway.

L7 ANSWER 2 OF 4 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All
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AN 2007484110 EMBASE
TI Involvement of RelB in aryl hydrocarbon receptor-mediated
induction of
chemokines.
AU Vogel, Christoph F.A. (correspondence); Sciullo, Eric;
Matsumura, Fumio
CS Department of Environmental Toxicology, University of
California, Davis,
One Shields Avenue, Davis, CA 95616, United States.
cfvogel@ucdavis.edu
SO Biochemical and Biophysical Research Communications, (23 Nov
2007) Vol.
363, No. 3, pp. 722-726.
Refs: 16
ISSN: 0006-291X; E-ISSN: 1090-2104 CODEN: BBRCA9
PUI S 0006-291X(07)01993-6
CY United States
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
LA English
SL English

ED Entered STN: 30 Oct 2007
Last Updated on STN: 30 Oct 2007
AB 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is a well-known immunotoxic
compound affecting the expression of inflammatory genes. We found that
TCDD induces the expression of the B-cell activating factor of the tumor
necrosis factor family (BAFF), B-lymphocyte chemoattractant (BLC), CC-chemokine ligand 1 (CCL1), and the transcription factor interferon
γ responsive factor (IFR3) in U937 macrophages in an aryl
hydrocarbon receptor- (AhR) and RelB-dependent manner. The induction was
associated with increased binding activity of an AhR/RelB complex without
participation of ARNT to a NF-.kappa.B
element that is recognized by the NF-.kappa.B
subunit RelB and localized on promoters of the cytokine and chemokine
genes BAFF, BLC, CCL1, and the transcription factor IRF3. The
interaction of AhR with RelB binding on a novel type of NF-.
kappa.B binding site represents a new regulatory
function of the AhR. .COPYRGT. 2007 Elsevier Inc. All rights reserved.

L7 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:324285 CAPLUS
DN 142:385993
TI Inhibitors of the IκB protein kinase α signal transduction
pathway for therapeutic regulation of gene expression
IN Karin, Michael; Bonizzi, Giussepina; Bebien, Magali
PA The Regents of the University of California, USA
SO PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005033284	A2	20050414	WO 2004-US32246	20040929
WO 2005033284	A3	20050707		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,

NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG
US 20080280286 A1 20081113 US 2008-574333 20080721
PRAI US 2003-508349P P 20031001
WO 2004-US32246 W 20040929
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 142:385993
AB Oligonucleotides that bind IκB kinase α (IKKα) that block its ability to induce cytokine-mediated gene expression are described for therapeutic use. Oligonucleotides that block the activation and interactions of the downstream transcription factors RelA and RelB. Expts. identifying the role of IKKα in the induction of chemokine gene expression in stromal cells are reported.

L7 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:717163 CAPLUS
DN 137:380824
TI Dynamic changes in histone H3 Lys 9 methylation occurring at tightly regulated inducible inflammatory genes
AU Saccani, Simona; Natoli, Gioacchino
CS Institute for Research in Biomedicine, Bellinzona, CH6501, Switz.
SO Genes & Development (2002), 16(17), 2219-2224
CODEN: GEDEEP; ISSN: 0890-9369
PB Cold Spring Harbor Laboratory Press
DT Journal
LA English
AB Methylation of histone H3 at Lys 9 is causally linked to formation of heterochromatin and to long-term transcriptional repression. We report an unexpected pattern of H3 Lys 9 methylation occurring at a subset of inducible inflammatory genes. This pattern is characterized by relatively low constitutive levels of H3 Lys 9 methylation that are erased upon

activation and restored concurrently with post-induction transcriptional repression. Changes in H3 Lys 9 methylation strongly correlate with RNA polymerase II recruitment and release. In particular, remethylation correlates with RNApolII release more strongly than does histone deacetylation. We propose that, by generating a window of time in which transcription is permitted, dynamic modulation of H3 Lys 9 methylation adds an addnl. regulatory level to transcriptional activation of tightly controlled inducible genes.
OSC.G 84 THERE ARE 84 CAPLUS RECORDS THAT CITE THIS RECORD (84 CITINGS)
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	44.10	78.07

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.70	

-3.40

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=> FIL BIOSIS CAPLUS EMBASE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.14	78.21

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	

-3.40

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=> d his

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     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:15:37 ON 20 APR 2010
L1            3 S (BLC OR ELC) (3A) PROMOTER
L2            3 DUP REM L1 (0 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:22:26 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:23:18 ON 20 APR 2010
L3            1 S NF KAPP B
L4       113335 S NF KAPPA B
L5           36 S L4 AND (BLC OR ELC)
L6            5 S L5 AND PROMOTER
L7            4 DUP REM L6 (1 DUPLICATE REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:28:53 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:29:56 ON 20 APR 2010

=> s CCL21 or CXCL13
L8         2987 CCL21 OR CXCL13

=> s l8 (3a) promoter
L9           11 L8 (3A) PROMOTER

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10           5 DUP REM L9 (6 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 5 ANSWERS - CONTINUE? Y/(N):y

L10  ANSWER 1 OF 5  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All
rights
     reserved on STN
AN   0019807029  EMBASE
CP   MEDLINE® is the source for the citation and abstract of this
record.
TI   New putative control elements in the promoter OF CXCL13
     chemokine gene, a target of alternative NF-kappaB pathway.
AU   Britanova, L.V. (correspondence); Kuprash, D.V.
SO   Molekuliarnaia biologiia, (2009 Jul-Aug) Vol. 43, No. 4, pp.
657-665.
     ISSN: 0026-8984
```

CY Russian Federation
DT Journal; Article
FS MEDLINE
LA Russian
ED Entered STN: 13 Apr 2010
Last Updated on STN: 13 Apr 2010
AB We searched the proximal promoter region of CXCL13/BLC chemokine gene for new putative control elements, including potential NF-kappaB binding sites. Using electrophoretic mobility shift assay and reporter gene analysis we identified two new promoter elements. The first element contains Rel/NF-kappaB binding site and seems to participate in inducible gene expression, while another site binds transcription factor Sp1 and is critical for basic transcription. It is the first indication that alternative NF-kappaB pathway target genes are probably cooperatively controlled by factors Rel/NF-kappaB and Sp1. Identification of a functional Sp1 site in the promoter of a target gene of alternative NF-kappaB pathway will be useful for investigation of molecular mechanisms and signal pathwaysinvolved.

L10 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2009:1037606 CAPLUS
TI New putative control elements in the promoter of the gene for the CXCL13 chemokine, a target of the alternative NF-κB pathway
AU Britanova, L. V.; Kuprash, D. V.
CS Engelgardt Institute of Molecular Biology, Russian Academy of Sciences, Moscow, 119991, Russia
SO Molecular Biology (Moscow, Russian Federation, English Edition) (2009), 43(4), 604-611
CODEN: MOLBBJ; ISSN: 0026-8933
PB Pleiades Publishing, Ltd.
DT Journal
LA English
AB The proximal promoter region of the gene for the CXCL13/BLC chemokine has been studied by electrophoretic mobility shift assay and reporter gene anal. in order to detect new control elements, in particular, NF-κB binding sites. Two new putative control elements have been identified.

One of them contains a Rel/NF-κB binding site and seems to participate in inducible gene expression. The other is an Sp1 factor binding site, essential for basal transcription. It is the first time that such a site is found in the promoter of a target gene of the alternative NF-κB pathway. This finding indicates that genes under the control of the alternative NF-κB pathway can be cooperatively regulated by Rel/NF-κB and Sp1. Our results will add to the understanding of the signaling pathways that govern the expression of genes controlled by the alternative NF-κB pathway.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 1
AN 2007:187927 BIOSIS
DN PREV200700189352
TI TNF receptor-associated factor 2-dependent canonical pathway is crucial for the development of Peyer's patches.
AU Piao, Jiang-Hu; Yoshida, Hisahiro; Yeh, Wen-Chen; Doi, Takahiro; Xue, Xin; Yagita, Hideo; Okumura, Ko; Nakano, Hiroyasu [Reprint Author]
CS Juntendo Univ, Sch Med, Dept Immunol, Bunkyo Ku, 2-1-1 Hongo, Tokyo 1138421, Japan
hnakano@med.juntendo.ac.jp
SO Journal of Immunology, (FEB 15 2007) Vol. 178, No. 4, pp. 2272-2277.
CODEN: JOIMA3. ISSN: 0022-1767.
DT Article
LA English
ED Entered STN: 14 Mar 2007
Last Updated on STN: 14 Mar 2007
AB Activation of the noncanonical pathway through the interaction of lymphotoxin (LT)-alpha(1)beta(2) and LT-beta R is essential for the development of secondary lymphoid organs including lymph nodes (LN) and Peyer's patches (PP). Although TNFR-associated factor (TRAF) 2 and TRAF5 were identified as signal transducers for the LT-OR, roles for TRAF2 and TRAF5 in the development of secondary lymphoid organs remain obscure. In this study, we show that PP but not mesenteric LN development is severely

impaired in traj2(-/-) and traf2(-/-)traf5(-/-) mice.
Development of
VCAM-1(+) and ICAM-1(+) mesenchymal cells and expression of
CXCL13, a
crucial chemokine for the development of PP, are severely
impaired in PP
anlagen in the intestines of traj2(-/-) mice. Surprisingly,
TNF-alpha
stimulation potently up-regulates cxcl13 mRNA expression in
wild-type
murine embryonic fibroblasts, which is impaired in trq/2(-/-) and
relA(-/-) murine embryonic fibroblasts. Moreover, RelA is
recruited to
the promoter of cxcl13 gene upon TNF-alpha stimulation
and PP development is impaired in TNFR type 1 (tnfr1)(-/-) mice.
These
results underscore a crucial role for the
TNFR1-TRAF2-RelA-dependent
canonical pathway in the development of PP through up-regulation
of cxcl13
mRNA.

L10 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson
Corporation on STN
DUPLICATE 2
AN 2007:463276 BIOSIS
DN PREV200700463443
TI Characterization of the CCL21-mediated melanoma-specific immune
responses
and in situ melanoma eradication.
AU Novak, Laura; Igoucheva, Olga; Cho, Stephanie; Alexeev, Vitali
[Reprint
Author]
CS Thomas Jefferson Univ, Jefferson Med Coll, Dept Dermatol and
Cutaneous
Biol, 233 S 10th St,BLSB,Room 326, Philadelphia, PA 19107 USA
vitali.alexeev@jefferson.edu
SO Molecular Cancer Therapeutics, (JUN 2007) Vol. 6, No. 6, pp.
1755-1764.
ISSN: 1535-7163.
DT Article
LA English
OS GenBank-MMU88322; EMBL-MMU88322; DDBJ-MMU88322
ED Entered STN: 29 Aug 2007
Last Updated on STN: 29 Aug 2007
AB Previous studies have shown that secondary lymphoid chemokine,
CCL21, can
be used for modulation of tumor-specific immune responses.
Here, using
B16F0 melanoma cells stably expressing CCL21 under the control of
cytomegalovirus and ubiquitin promoters, we showed that
CCL21-activated

immune responses depend on the amount of melanoma-derived chemokine,
which, in turn, depends on the strength of the promoter. We showed that
ubiquitin promoter-driven expression of CCL21 enabled
massive infiltration of tumors with CD4(+)CD25(-), CD8(+) T lymphocytes,
and CD11c(+) dendritic cells, and consequent activation of cellular and
humoral immune responses sufficient for complete rejection of
CCL21-positive melanomas within 3 weeks in all tumor-inoculated mice.
Mice that rejected CCL21-positive tumors acquired protective immunity
against melanoma, which was transferable to naive mice via splenocytes and
central memory T cells. Moreover, melanoma-derived CCL21 facilitated
immune-mediated remission of preestablished, distant wild-type melanomas.
Overall, these results suggest that elevated levels of tumor-derived CCL21
are required for the activation of strong melanoma-specific immune
responses and generation of protective immunologic memory. They also open
new perspectives for the development of novel vaccination strategies
against melanoma, which use intratumoral delivery of the optimized
CCL21-encoding vectors in conjunction with DNA-based vaccines.

L10 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3
AN 2005:53628 BIOSIS
DN PREV200500053206
TI A novel model for lymphocytic infiltration of the thyroid gland generated
by transgenic expression of the CC chemokine CCL21.
AU Martin, Andrea P.; Coronel, Elizabeth C.; Sano, Gen-ichiro; Chen, Shu-g;
Vassileva, Galya; Canasto-Chibuque, Claudia; Sedgwick, Jonathon D.;
Frenette, Paul S.; Lipp, Martin; Furtado, Glaucia C.; Lira, Sergio A.
[Reprint Author]
CS Immunobiol Ctr, Mt Sinai Sch Med, 1425 Madison Ave,Box 1630, New York, NY,
10029, USA
sergio.lira@mssm.edu
SO Journal of Immunology, (October 15 2004) Vol. 173, No. 8, pp. 4791-4798.

print.
ISSN: 0022-1767 (ISSN print).
DT Article
LA English
ED Entered STN: 3 Feb 2005
Last Updated on STN: 3 Feb 2005
AB Lymphocytic infiltrates and lymphoid follicles with germinal centers are
often detected in autoimmune thyroid disease (AITD), but the mechanisms
underlying lymphocyte entry and organization in the thyroid remain
unknown. We tested the hypothesis that CCL21, a chemokine that regulates
homeostatic lymphocyte tracking, and whose expression has been detected in
AITD, is involved in the migration of lymphocytes to the thyroid. We show
that transgenic mice expressing CCL21 from the thyroglobulin
promoter (TGCCL21 mice) have significant lymphocytic infiltrates,
which are topologically segregated into B and T cell areas. Although high
endothelial venules expressing peripheral lymph node addressin were
frequently observed in the thyroid tissue, lymphocyte recruitment was
independent of L-selectin or lymphotoxin-a but required CCR7 expression.
Taken together, these results indicate that CCL21 is sufficient to drive
lymphocyte recruitment to the thyroid, suggest that CCL21 is involved in
AITD pathogenesis, and establish TGCCL21 transgenic mice as a novel model
to study the formation and function of lymphoid follicles in the thyroid.

=> d his

(FILE 'HOME' ENTERED AT 12:06:56 ON 20 APR 2010)

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:15:37 ON 20 APR 2010
L1 3 S (BLC OR ELC) (3A) PROMOTER
L2 3 DUP REM L1 (0 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 12:22:26 ON 20 APR 2010

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:23:18 ON 20 APR 2010
L3 1 S NF KAPP B
L4 113335 S NF KAPPA B
L5 36 S L4 AND (BLC OR ELC)

```
L6             5 S L5 AND PROMOTER
L7             4 DUP REM L6 (1 DUPLICATE REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:28:53 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:29:56 ON 20 APR 2010
L8          2987 S CCL21 OR CXCL13
L9            11 S L8 (3A) PROMOTER
L10            5 DUP REM L9 (6 DUPLICATES REMOVED)

=> s l4 and l8
L11         110 L4 AND L8

=> s l11 and promoter
L12           5 L11 AND PROMOTER

=> dup rem l5
PROCESSING COMPLETED FOR L5
L13           22 DUP REM L5 (14 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 22 ANSWERS - CONTINUE? Y/(N):y

L13  ANSWER 1 OF 22  BIOSIS  COPYRIGHT (c) 2010 The Thomson
Corporation  on STN
     DUPLICATE 1
AN   2010:175759  BIOSIS
DN   PREV201000175759
TI   SLC/CCR7 Stimulates the Proliferation of BMDCs by the pNF-kappa
B p65
     Pathway.
AU   Zhou, Shuang; Li, Rilun; Qin, Jie; Zhong, Cuiping; Liang,
Chunmin [Reprint
     Author]
CS   Fudan Univ, Shanghai Med Coll, Dept Anat Histol and Embryol, 138
Yixueyuan
     Rd, Shanghai 200032, Peoples R China
     cpzhong@shmu.edu.cn; cmliang@fudan.edu.cn
SO   Anatomical Record, (JAN 2010) Vol. 293, No. 1, pp. 48-54.
     ISSN: 1932-8486. E-ISSN: 1932-8494.
DT   Article
LA   English
ED   Entered STN: 31 Mar 2010
     Last Updated on STN: 31 Mar 2010
AB   The chemokine receptor CCR7 is highly expressed in dendritic
cells (DCs),
     T cells, and other immune effector cells.  One of the
high-affinity ligand
     that can bind to CCR7 is the secondary lymphoid tissue chemokine
(SLC).
     The SLC/CCR7 axis plays an important role in the immune system
by inducing
```

the chemotaxis and migration of immune effector cells. In this study, we
examined the effect of SLC at different concentrations (0, 50, 100, 200,
300, and 400 ng/mL) on the proliferation of bone-marrow-derived dendritic
cells (BMDCs). ELC (CCL19), another high-affinity ligand for
CCR7, was used as the control at the same time. We found that SLC
directly stimulated the proliferation of BMDCs and enhanced the
antigen-presenting function and CCR7 expression. Western blot analysis
showed that pNF-kappa Bp65 was involved in this mechanism. We also found
that the NF-kappa B inhibitor PDTC could
specifically block the proliferation and CCR7 expression of BMDCs induced
by SLC or ELC (200 ng/mL). The results suggested that there
were cross-talk signals between the chemotaxis and proliferation of BMDCs
involving the SLC/CCR7 axis. Anat Rec, 293:48-54, 2010. (C) 2009
Wiley-Liss, Inc.

L13 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2010:314782 CAPLUS
TI Signaling mechanism of NO-induced increase in cardiac tolerance to
ischemia-reperfusion
AU Maslov, L. N.; Kolar, F.; Barsakh, E. I.
CS Scientific-Research Institute of Cardiology of Siberian Branch, RAMS,
Tomsk, Russia
SO Rossiiskii Fiziologicheskii Zhurnal imeni I. M. Sechenova (2009), 95(11),
1175-1189
CODEN: RFZSFY; ISSN: 1029-595X
PB Sankt-Peterburgskaya Izdatel'skaya Firma RAN "Nauka"
DT Journal
LA Russian
AB In the review it is analyzes published data on the signaling mechanism of
cardioprotective impact of nitric oxide. It was shown that nitric oxide
exhibited a rapid and a delayed cardioprotective effects. In the rapid
effect, endothelial NO-synthase (NOS) is involved was involved as well as
guanylate cyclase, cGMP, kinase G, kinase C, PI3-kinase, Akt-kinase,
mitochondrial ATP-sensitive K+-channel, reactive oxygen species, MPT-pore.
Delayed cardioprotective effect of NOS required synthesis of proteins de

novo. In this process, transcription factors NF-.kappa
.B, STAT1/3, HIF-1 are involved. Some published data state that
peroxynitrite, cGMP, kinase G, kinase C, Src kinase, p38 kinase,
ERX-kinase can be involved in delayed cardioprotective effect of
NOS. The
cardioprotective impact of nitric oxide was shown to depend on
enhancement
in expression of NOS, cyclooxygenase-2 and Blc-2 protein which
inhibits MPT-pore.

L13 ANSWER 3 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson
Corporation on STN
DUPLICATE 2
AN 2009:551971 BIOSIS
DN PREV200900553074
TI New putative control elements in the promoter of the gene for
the CXCL13
chemokine, a target of the alternative NF-kappa
B pathway.
AU Britanova, L. V. [Reprint Author]; Kuprash, D. V.
CS Russian Acad Sci, VA Engelhardt Mol Biol Inst, Moscow 119991,
Russia
kuprash@eimb.ru
SO Molecular Biology (Moscow), (AUG 2009) Vol. 43, No. 4, pp.
604-611.
CODEN: MOLBBJ. ISSN: 0026-8933. E-ISSN: 1608-3245.
DT Article
LA English
ED Entered STN: 30 Sep 2009
Last Updated on STN: 30 Sep 2009
AB The proximal promoter region of the gene for the CXCL13/BLC
chemokine has been studied by electrophoretic mobility shift
assay and
reporter gene analysis in order to detect new control elements,
in
particular, NF-kappa B binding sites. Two
new putative control elements have been identified. One of them
contains
a Rel/NF-kappa B binding site and seems to
participate in inducible gene expression. The other is an Sp1
factor
binding site, essential for basal transcription. It is the
first time
that such a site is found in the promoter of a target gene of the
alternative NF-kappa B pathway. This
finding indicates that genes under the control of the alternative
NF-kappa B pathway can be cooperatively
regulated by Rel/NF-kappa B and Sp1. Our
results will add to the understanding of the signaling pathways
that
govern the expression of genes controlled by the alternative NF-
kappa B pathway.

L13 ANSWER 4 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3
AN 2008:426137 BIOSIS
DN PREV200800426136
TI Distinct effect of CD40 and TNF-signaling on the chemokine/chemokine
receptor expression and function of the human monocyte-derived dendritic
cells.
AU Xia, Yu; Dai, Jun; Lu, Peirong; Huang, Yong; Zhu, Yipei; Zhang, Xueguang
[Reprint Author]
CS Soochow Univ, Med Biotechnol Inst, 708 Renmin Rd, Suzhou 215007, Jiangsu,
Peoples R China
smbxuegz@public1.sz.js.cn
SO Cellular & Molecular Immunology, (APR 2008) Vol. 5, No. 2, pp. 121-131.
ISSN: 1672-7681.
DT Article
LA English
ED Entered STN: 6 Aug 2008
Last Updated on STN: 6 Aug 2008
AB A key and limiting step in the process of human monocyte-derived dendritic
cells (mDCs) for clinical use is their in vitro maturation and in vivo
migration. We previously observed that CD40 signal facilitated human mDC
growth and maturation. To further explore this process, mDCs generated
with GM-CSF and IL-4 were co-cultured with apoptotic tumor cells for 24
hours, followed by incubating with anti-CD40 monoclonal antibody or
TNF-alpha for 48 hours to generate mature DCs. The chemokine/chemokine
receptor expression and functions of mature DCs upon various stimuli were
determined. The expression of costimulatory molecules on apoptotic tumor
cell-loaded mature DCs co-cultured with either anti-CD40 antibody
(anti-CD40-DCs) or TNF-alpha (TNF-DCs) were up-regulated compared to
immature DCs, consistent with the abilities of these cytokine to drive DC
maturation in vitro. The mRNA levels of chemokines such as stromal
cell-derived factor-1 alpha (SDF-1 alpha), EBV-induced molecule 1 ligand

chemokine (ELC), and IFN inducible protein-10 (IP-10) in
anti-CD40 activated DO were increased and the dendritic
cell-specific
chemokine 1 (DC-CK1) was moderately up-regulated as compared
with other
mature DCs. The corresponding chemokine receptors CXCR4 and
CCR7 of
anti-CD40-DCs were significantly expressed. The CXCR3
expression on
activated T cells stimulated by anti-CD40-DCs was also increased.
Moreover, the anti-CD40-DCs had a stronger ability to stimulate
T cell
proliferation than any other DCs. The NF-kappa
B activity was much higher in anti-CD40-DCs than that of TNF-DCs.
These results offer further evidence of the importance of the
CD40 signal
in developing efficient human DC vaccines for cancer immune
therapy.

L13 ANSWER 5 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson
Corporation on STN
DUPLICATE 4
AN 2008:11118 BIOSIS
DN PREV200800002229
TI Involvement of RelB in aryl hydrocarbon receptor-mediated
induction of
chemokines.
AU Vogel, Christoph F. A. [Reprint Author]; Sciullo, Eric;
Matsumura, Fumio
CS Univ Calif Davis, Dept Environm Toxicol, 1 Shields Ave, Davis,
CA 95616
USA
cfvogel@ucdavis.edu
SO Biochemical and Biophysical Research Communications, (NOV 23
2007) Vol.
363, No. 3, pp. 722-726.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 12 Dec 2007
Last Updated on STN: 12 Dec 2007
AB 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is a well-known
immunotoxic
compound affecting the expression of inflammatory genes. We
found that
TCDD induces the expression of the B-cell activating factor of
the tumor
necrosis factor family (BAFF), B-lymphocyte chemoattractant (BLC
), CC-chemokine ligand I (CCL1), and the transcription factor
interferon
gamma responsive factor (IFR3) in U937 macrophages in an aryl
hydrocarbon

receptor- (AhR) and RelB-dependent manner. The induction was associated
with increased binding activity of an AhR/RelB complex without participation of ARNT to a NF-kappa B
element that is recognized by the NF-kappa B
subunit RelB and localized on promoters of the cytokine and chemokine
genes BAFF, BLC, CCL 1, and the transcription factor IRF3. The interaction of AhR with RelB binding on a novel type of NF-kappa B binding site represents a new regulatory
function of the AhR. (C) 2007 Elsevier Inc. All rights reserved.

L13 ANSWER 6 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 5
AN 2006:370141 BIOSIS
DN PREV200600369173
TI NF-kappa B-inducing kinase regulates
selected gene expression in the Nod2 signaling pathway.
AU Pan, Qilin; Kravchenko, Vladimir; Katz, Alex; Huang, Shuang; Ii, Masayuki;
Mathison, John C.; Kobayashi, Koichi; Flavell, Richard A.; Schreiber,
Robert D.; Goeddel, David; Ulevitch, Richard J. [Reprint Author]
CS Scripps Res Inst, Dept Immunol, 10550 N Torrey Pines Rd,IMM-12, La Jolla,
CA 92037 USA
ulevitch@scripps.edu
SO Infection and Immunity, (APR 2006) Vol. 74, No. 4, pp. 2121-2127.
CODEN: INFIBR. ISSN: 0019-9567.
DT Article
LA English
ED Entered STN: 26 Jul 2006
Last Updated on STN: 26 Jul 2006
AB The innate immune system surveys the extra- and intracellular environment
for the presence of microbes. Among the intracellular sensors is a
protein known as Nod2, a cytosolic protein containing a leucine-rich
repeat domain. Nod2 is believed to play a role in determining host
responses to invasive bacteria. A key element in upregulating host
defense involves activation of the NF-kappa B
pathway. It has been suggested through indirect studies that NF-kappa B inducing kinase, or NIK, may be involved in
Nod2 signaling. Here we have used macrophages derived from primary
explants of bone marrow from wild-type mice and mice that either bear a

mutation in NIK, rendering it inactive, or are derived from NIK-/- mice, in which the NIK gene has been deleted. We show that NIK binds to Nod2 and mediates induction of specific changes induced by the specific Nod2 activator, muramyl dipeptide, and that the role of NIK occurs in settings where both the Nod2 and TLR4 pathways are activated by their respective agonists. Specifically, we have linked NIK to the induction of the B-cell chemoattractant known as BLC and suggest that this chemokine may play a role in processes initiated by Nod2 activation that lead to improved host defense.

L13 ANSWER 7 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:729611 CAPLUS
DN 143:206465
TI Therapeutic and carrier molecules
IN Ferrante, Antonio; Rathjen, Deborah Ann
PA Peplin Biolipids Pty Ltd, Australia
SO PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005073164	A1	20050811	WO 2005-AU98	20050128

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,

RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE, SN, TD, TG
AU 2005209331 A1 20050811 AU 2005-209331 20050128
CA 2554735 A1 20050811 CA 2005-2554735 20050128
EP 1718602 A1 20061108 EP 2005-700130 20050128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS
CN 1934072 A 20070321 CN 2005-80008891 20050128
BR 2005007236 A 20070626 BR 2005-7236 20050128
JP 2007522118 T 20070809 JP 2006-549788 20050128
US 20090215895 A1 20090827 US 2009-588094 20090507
PRAI US 2004-540604P P 20040130
WO 2005-AU98 W 20050128
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 143:206465
AB The present invention relates generally to compds. comprising a hydrocarbon chain portion and more particular to compds. comprising chemical
derivatizations of the hydrocarbon chain which are useful therapeutic and
prophylactic mols. The present invention further provides compds. where
the hydrocarbon chain portion is a carrier mol. for functional groups,
moieties or agents. The present invention can include naturally including
polyunsatd. fatty acids as well as synthetic, modified or derivatized
polyunsatd. fatty acids. Furthermore. these polyunsatd. fatty acids can
be conjugated to amino acids, peptides or proteins. The compds. of the
present invention are particularly useful in the treatment and prophylaxis
of a range of conditions including cancers, protein kinase c(PKC)- or
NF.kappa.B-related- or -associated conditions,
cardiovascular conditions, pain, inflammatory conditions, vascular or
immunol. conditions such as diabetes, neurol. conditions and infection by
a range of viruses or prokaryotic or eukaryotic organisms. The present

invention further provides pharmaceutical compns. and methods of medical
treatment.
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 8 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:395470 CAPLUS
DN 142:442896
TI Methods for differentiating stem cells using a self-replicating
neocentromeric artificial chromosome with chromatin domains expressing
transgenes for gene therapy
IN Choo, Kong-Hong Andy; Wong, Lee Hwa; Saffery, Richard Eric
PA Murdoch Childrens Research Institute, Australia
SO PCT Int. Appl., 168 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005040391	A1	20050506	WO 2004-AU1469	20041025

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI AU 2003-905894 A 20031027
AB The present invention relates to the field of tissue engineering and

genetic manipulation of cells and to methods for generating tissue
suitable for use in repair, replacement, rejuvenation or augmentation
therapy. The present invention contemplates a method for genetically
manipulating a stem cell by introducing a nucleic acid mol. comprising a
centromere or neo-centromere into the stem cell, wherein the nucleic acid
mol. conveys genetic information which is capable of introducing to or
modifying a trait within the stem cell or progeny of the stem cell such as
but not limited to modulating the level of stem cell proliferation,
differentiation and/or self-renewal. The neo-centromere is devoid of
α-satellite repeat DNA. One aspect of the present invention
provides a stem cell comprising a self-replicating artificial chromosome
with a neo-centromere having centromeric chromatin domains comprising
expressible genetic material which modifies or introduces at least one
trait in said stem cell. Microarray gene expression profiles were
conducted for human 10q25 centromeric region. The engineered stem cells
may also be re-programmed, for example, to direct the cells down a
different cell lineage.
OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 9 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2005:324285 CAPLUS
DN 142:385993
TI Inhibitors of the IκB protein kinase α signal transduction
pathway for therapeutic regulation of gene expression
IN Karin, Michael; Bonizzi, Giussepina; Bebien, Magali
PA The Regents of the University of California, USA
SO PCT Int. Appl., 128 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI WO 2005033284 A2 20050414 WO 2004-US32246
20040929
WO 2005033284 A3 20050707
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ,
CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI,
GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,
KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ,
NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,
SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA,
ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM,
ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ,
DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT,
RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE,
SN, TD, TG
US 20080280286 A1 20081113 US 2008-574333
20080721
PRAI US 2003-508349P P 20031001
WO 2004-US32246 W 20040929
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OS MARPAT 142:385993
AB Oligonucleotides that bind IκB kinase α (IKKα) that
block its ability to induce cytokine-mediated gene expression are
described for therapeutic use. Oligonucleotides that block the
activation
and interactions of the downstream transcription factors RelA
and RelB.
Expts. identifying the role of IKKα in the induction of
chemokine
gene expression in stromal cells are reported.

L13 ANSWER 10 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson
Corporation on
STN
AN 2006:209029 BIOSIS
DN PREV200600210758
TI Helicobacter pylori contributes to lymphocyte infiltration and
anti-apoptosis via NF-kappab alternative pathway.
AU Ohmae, Tomoya; Hirata, Yoshihiro; Maeda, Shin; Shibata, Wataru;
Yanai,
Ayako; Ogura, Keiji; Yamaji, Yutaka; Okamoto, Makoto; Yoshida,
Haruhiko;
Kawabe, Takao; Omata, Masao

SO Gastroenterology, (APR 2005) Vol. 128, No. 4, Suppl. 2, pp. A350.
Meeting Info.: Annual Meeting of the American-Gastroenterological-Association/Digestive-Disease-Week. Chicago, IL, USA. May 14 -19, 2005. Amer Gastroenterol Assoc.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 29 Mar 2006
Last Updated on STN: 29 Mar 2006
AB Background and aim: Helicobacter pylori infection is known as a major cause of chronic active gastritis, accompanied with lymphocytic infiltration. We have reported that the bacterium activates NF-kB via both classical and alternative pathway in lymphocyte in vitro, Although the activation of classical pathway is reported to induce anti-apoptosis, the consequence of the activation of alternative pathway is not fully understood. in this study, we have examined the effect of alternative pathway activation on cell proliferation and apoptosis in vitro, The activation of alternative pathway was also investigated in vivo.Methods: The effect of the activation of NF-kB alternative pathway by H. pylon on apoptosis was analyzed in IM-9, human lymphoblastoid cell line. Some cells were pretreated with siRNAs for IKKa, or NF-kB2/p100, and then stimulated with H. pylon cells (MOI 100). The apoptosis of the human cells was analyzed by cell death detection ELISA. The cell proliferation was examined by BrdU ELISA. The localization of NF-kB2/p100 in human gastric mucosa was also investigated by immunohistochemistry in patients with and without H. Pylon infection. The expression of blc, etc and sdf-1-al the target genes of the NF-kB alternative pathway in gastric mucosa was analyzed with RT-PCR.Results: H. pylori enhanced apoptosis of IM-9 cells 1.8 + -0.4-fold in untreated cells. This proapoptotic effect of H. Pylon was further enhanced 2.1-fold by IKKa and 2.2-fold by NFkB2/p100 silencing ($p<0.05$ for each siRNA compared with

control siRNA), suggesting that alternative pathway was involved in anti-apoptotic response. Cell proliferation induced by H. Pylon was not markedly affected by IKKa or NF-kB2/p100 siRNA. In H. pylon-infected mucosa, NF-kB2/p100 and p52 were immunohistochemically detected in both cytoplasm and nucleus of lymphocytes but nowhere in epithelial cells. The mRNA expression of blc, etc, and sdf-1-a in the gastric tissue was very low in uninfected mucosa, while markedly up-regulated in H. pylon-infected mucosa.Conclusion: In H. pylori-infected tissue, NF-kB alternative pathway was found to be activated only in lymphocytes. Our results showed that H. Pylon up-regulates chemokine gene expression and induces anti-apoptosis both in vivo and in vitro. The activation of NF-kB alternative pathway may promote lymphocyte infiltration into gastric epithelium and may allow lymphocytes to acquire malignant potential.

L13 ANSWER 11 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 2006:78291 BIOSIS

DN PREV200600085032

TI Helicobacter pylori activates Nf-kappa B via both classical and alternative pathway in murine and human peripheral blood mononuclear cells.

AU Ohmae, Tomoya; Hirata, Yoshihiro; Maeda, Shin; Shibata, Watarn; Yanai, Ayako; Ogura, Keiji; Yoshida, Haruhiko; Kawabe, Takao; Omata, Masao

SO Gastroenterology, (APR 2004) Vol. 126, No. 4, Suppl. 2, pp. A405. Meeting Info.: Digestive Disease Week/105th Annual Meeting of the American-Gastroenterological-Association. New Orleans, LA, USA. May 16 -20, 2004. Amer Gastroenterol Assoc. CODEN: GASTAB. ISSN: 0016-5085.

DT Conference; (Meeting) Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 25 Jan 2006 Last Updated on STN: 25 Jan 2006

AB Background and aim: Although gastric Mucosa-associated lymphoid tissue

(MALT) lymphoma is associated with chronic infection of Helicobacter pylon (H. pylon). it is not clear how IT pylori contributes to the development of MALT lymphoma. Recently, especialy in B lymphocytes, the alternative pathway for NF-kappa B activation, which includes IKK alpha and NF-kappa B2/p52, has been reported to contribute to B cell development, survival, attenuation of apoptosis, and even proliferation. In this study, we analyzed whether H, Pylon induced NF-kappa B activation through both classical and alternative pathways in murine and human peripheral blood mononuclear cells. The role of cag PAI for NF-kappa B activation in lymphocytes was also examined. Methods: Murine, splenic B cells and peripheral blood mononuclear cells from human healthy volunteer were cultured with or without H. pylon cells (TN2 and its knockout mutant Delta cagE). After several hours, the cells were harvested and the total cellular lysates were prepared immediately. Western blot analysis was performed to detect p-I kappa B alpha, I kappa B alpha, and NF-kappa B2 (p52 and its precursor p100). Total cellular RNA was also extracted. The expression of blc, elc, or sdf-1-alpha was analyzed by RT-PCR. Results: In murine splenic B cells and human peripheral blood mononuclear cells, H. pylori infection induced I kappa B alpha phosphorylation as seen in gastric epitherial cells. In addition, NF-kappa B 2/p52 was also increased by H. Pylon in Western blot analysis. The mRNA expression of blc, elc, or sdf-1-alpha, all known as NF-kappa B2/p52 target genes, was upregulated by H. pylon infection after 8 hours. TN2 Delta cagE induced I kappa B alpha phosphorylation and NF-kappa B2/p52 production to the similar extent as the wild type did. Conclusion: In both murine splenic B cells and human peripheral blood mononuclear cells, H. pylori activated the alternative NF-kappa B signaling pathway, related to NF-kappa B2/p52, as well as the classical pathway involving I kappa B alpha. H. Pylon cag PAI does not seem to

have any roles for the NF-kappa B activation
of lymphocytes. These results support the idea that H. pylon stimulates B
cell proliferation through NF-kappa B
pathways and may promote MALT lymphomas by direct interaction.

L13 ANSWER 12 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN DUPLICATE 6
AN 2004:147630 BIOSIS
DN PREV200400151114
TI Epstein-Barr virus latent infection membrane protein 1 TRAF-binding site
induces NIK/IKKalpha-dependent noncanonical NF-kappaB activation.
AU Luftig, Micah; Yasui, Teruhito; Soni, Vishal; Kang, Myung-soo; Jacobson,
Nils; Cahir-McFarland, Ellen; Seed, Brian; Kieff, Elliott [Reprint Author]
CS Channing Laboratory, Brigham and Women's Hospital, 181 Longwood Avenue,
8th Floor, Boston, MA, 02115, USA
ekieff@rics.bwh.harvard.edu
SO Proceedings of the National Academy of Sciences of the United States of
America, (January 6 2004) Vol. 101, No. 1, pp. 141-146. print.
ISSN: 0027-8424 (ISSN print).
DT Article
LA English
ED Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004
AB Epstein-Barr virus (EBV) latent infection membrane protein 1
(LMP1)-induced NF-kappaB activation is important for infected cell
survival. LMP1 activates NF-kappaB, in part, by engaging tumor necrosis
factor (TNF) receptor-associated factors (TRAFs), which also mediate
NF-kappaB activation from LTbetaR and CD40. LTbetaR and CD40 activation
of p100/NF-kappaB2 is now known to be NIK/IKKalpha-dependent and
IKKbeta/IKKgamma independent. In the experiments described here, we found
that EBV LMP1 induced p100/NF-kappaB2 processing in human lymphoblasts and
HEK293 cells. LMP1-induced p100 processing was NIK/IKKalpha dependent and
IKKbeta/IKKgamma independent. Furthermore, the LMP1 TRAF-binding site was
required for p100 processing and p52 nuclear localization, whereas the
LMP1 death domain-binding site was not. Moreover, the LMP1 TRAF-binding

site preferentially caused RelB nuclear accumulation. In murine embryo
fibroblasts (MEFs), IKKbeta was essential for LMP1 up-regulation of
macrophage inflammatory protein (MIP)-2, TNFalpha, I-TAC, ELC,
MIG, and CXCR4 RNAs. Interestingly, in IKKalpha knockout MEFs, LMP1
hyperinduced MIP-2, TNFalpha, and I-TAC expression, consistent with a role
for IKKalpha in down-modulating canonical IKKbeta activation or its
effects. In contrast, LMP1 failed to up-regulate CXCR4 and MIG RNA in
IKKalpha knockout MEFs, indicating a dependence on noncanonical IKKalpha
activation. Furthermore, LMP1 up-regulation of MIP-2 RNA in MEFs was both
IKKbeta- and IKKgamma-dependent, whereas LMP1 up-regulation of MIG and
I-TAC RNA was fully IKKgamma independent. Thus, LMP1 induces typical
canonical IKKbeta/IKKgamma-dependent, atypical canonical
IKKbeta-dependent/IKKgamma-independent, and noncanoni-cal
NIK/IKKalpha-dependent NF-kappaB activations; NIK/IKKalpha-dependent
NF-kappaB activation is principally mediated by the LMP1 TRAF-binding
site.

L13 ANSWER 13 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2003:373862 CAPLUS
DN 138:380364
TI A nucleic acid array of genes associated with disease responses in
macrophages and their use in the diagnosis of disease
IN Stuhlmueller, Bruno; Haeupl, Thomas
PA Oligene G.m.b.H., Germany
SO Eur. Pat. Appl., 180 pp.
CODEN: EPXXDW
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1310567	A2	20030514	EP 2002-90348	20021002
EP 1310567	A3	20040225		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

DE 10155600 A1 20030522 DE 2001-10155600 20011109
DE 10155600 B4 20090827
US 20050037344 A1 20050217 US 2002-278698 20021023
PRAI DE 2001-10155600 A 20011109
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB An array of ≈250 genes that show differential expression in macrophages in health and immune disorders is described for use in the diagnosis and monitoring of macrophage associated immune disorders and in screening of drugs.
OSC.G 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 14 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2004:154844 BIOSIS
DN PREV200400148381
TI Imatinib mesylate (STI571) can act on non-malignant CD34+ peripheral blood progenitor cells by affecting their development into dendritic cells.
AU Appel, Silke [Reprint Author]; Boehmler, Andreas M. [Reprint Author]; Gruenebach, Frank [Reprint Author]; Mueller, Martin R. [Reprint Author]; Rupf, Anette [Reprint Author]; Weck, Markus M. [Reprint Author]; Hartmann, Ulrike [Reprint Author]; Reichardt, Volker L. [Reprint Author]; Kanz, Lothar [Reprint Author]; Brummendorf, Tim H. [Reprint Author]; Brossart, Peter [Reprint Author]
CS Hematology, Oncology and Immunology, Internal Medicine II, University of Tuebingen, Tuebingen, Germany
SO Blood, (November 16 2003) Vol. 102, No. 11, pp. 826a. print. Meeting Info.: 45th Annual Meeting of the American Society of Hematology. San Diego, CA, USA. December 06-09, 2003. American Society of Hematology. CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting) Conference; (Meeting Poster) Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Mar 2004

Last Updated on STN: 17 Mar 2004

AB Imatinib mesylate (STI571; Glivec) is a competitive Bcr-Abl tyrosine kinase inhibitor and has yielded encouraging results in treatment of chronic myelogenous leukemia (CML) and gastrointestinal stroma tumors. Apart from inhibition of the Abl protein tyrosine kinases, it also shows activity against PDGF-R, c-Kit, ARG and their fusion proteins while sparing other kinases. In vitro studies have revealed that imatinib mesylate can inhibit growth of cell lines and primitive malignant progenitor cells in CML expressing Bcr-Abl. However, little is known about the effects of imatinib mesylate on non-malignant hematopoietic cells. Since the ligand of c-Kit, stem cell factor (SCF), has been shown to play an important role in development of dendritic cells (DC), we here explored a potential effect of STI571 on the development of mobilized human CD34+ peripheral blood progenitor cells into DC. In our study we demonstrate that in vitro exposure of mobilized human CD34+ progenitors to therapeutic concentrations of imatinib mesylate (1-5)muM) inhibits their differentiation into dendritic cells. DC obtained after 10-16 days of culture in the presence of STB71 showed concentration dependent reduced expression levels of CD1a and co-stimulatory molecules like CD80 and CD40 without affecting their morphology or viability. Expression analyses of chemokines known to be important for DC function by RT-PCR revealed an increased expression of MIP-1a whereas no differences in the expression of TARC and the chemokine receptor CCR6 were observed. In contrast, mRNA levels of ELC (CCL19) and the corresponding receptor CCR7 were reduced in the presence of imatinib mesylate. Furthermore, exposure to STI571 inhibited CD40 ligand induced activation of generated DC and the initiation of primary CTL responses. To determine the possible role of c-Kit in the observed inhibition of DC development, we incubated CD34+

cells with blocking antibodies against SCF and its receptor c-Kit.
However, no effect on DC development could be detected indicating that
imatinib mesylate acts by inhibition of other tyrosine kinases. The
effects of imatinib mesylate were accompanied by downregulation of nuclear
localized RelB protein which has been shown to be important for DC
differentiation and function. Interestingly, there was no reduction in
the expression of c-Rel or RelA proteins, other members of the NF-kappaB
family. Our results demonstrate that imatinib mesylate can act on normal
hematopoietic cells and inhibits the differentiation and function of DC by
interfering with the NF-kappaB signal transduction pathway.

L13 ANSWER 15 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2003:120036 CAPLUS
DN 138:236622
TI RelB in secondary lymphoid organ development: differential regulation by
lymphotoxin and tumor necrosis factor signaling pathways
AU Yilmaz, Z. Buket
CS Institut fuer Toxikologie und Genetik, Germany
SO Wissenschaftliche Berichte - Forschungszentrum Karlsruhe (2002), FZKA
6793, i-xv, 1-117
CODEN: WBFKF5; ISSN: 0947-8620
DT Report
LA English
AB Primary lymphoid organs are the major sites of lymphopoiesis where
lymphocytes proliferate and mature into functional but naive cells.
Secondary lymphoid organs are sites where these lymphocytes encounter
antigens and elicit immune responses. RelB is a member of the Rel/
NF-.kappa.B family of inducible dimeric
transcription factors. RelB is abundantly expressed in secondary lymphoid
organs, such as spleen, lymph nodes, and Peyer's patches (PP).
RelB-deficient mice have improper spleen structure and lack organizing
centers for PPs, defects that can not be restored by the adoptive transfer
of wild-type bone marrow cells. The work presented here revealed a reduction

in expression of the homing chemokines B lymphocyte chemoattractant (BLC) and secondary lymphoid organ chemokine (SLC) in RelB-deficient spleen, suggesting a role for RelB in proper expression of chemokines by splenic stromal cells. Moreover, interleukin-7 (IL-7)-induced expression of lymphotoxin (LT) in intestinal cells, a crucial step in early PP development, was not impaired in RelB-deficient embryos, suggesting functional hematopoietic inducers and a defect in LTβ receptor (LTβR) expressing stromal responders. Activation of LTβR signaling in fibroblasts resulted in the specific induction of p52-RelB heterodimers, while tumor necrosis factor (TNF) induced classical p50-RelA NF-.kappa.B complexes. LTβR-induced RelB nuclear translocation and DNA binding of p52-RelB heterodimers required the degradation of the inhibitory p52 precursor, p100, which was dependent on the IκB kinase (IKK) complex subunit IKKα, but not on IKKβ or IKKγ. In contrast to LTβR signaling, TNFR signaling increased p100 and RelB levels both in cytoplasm and nucleus and RelB was bound to p100 in both compartments. Despite the abundant presence of RelB in the nucleus, RelB DNA binding was almost undetectable in TNF treated fibroblasts. Forced expression of p50 and p52 could not rescue the lack of DNA binding. In contrast, RelB DNA binding increased in cells lacking the C-terminus of p100, but not of p105, strongly suggesting that it is the specific inhibitory function of the C-terminal domain of p100, rather than the lack of the heterodimerization partner, which prevents RelB DNA binding in TNF-stimulated fibroblasts. Thus, RelB and p52 in stromal cells could function in the proper development of the spleen by regulating the expression of chemokines such as BLC. Furthermore, generation of p52-RelB heterodimers by the LTβR pathway involving p100 degradation, appears to be a critical step in the formation of PP anlage.

RE.CNT 118 THERE ARE 118 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 16 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:717163 CAPLUS
DN 137:380824
TI Dynamic changes in histone H3 Lys 9 methylation occurring at tightly
regulated inducible inflammatory genes
AU Saccani, Simona; Natoli, Gioacchino
CS Institute for Research in Biomedicine, Bellinzona, CH6501, Switz.
SO Genes & Development (2002), 16(17), 2219-2224
CODEN: GEDEEP; ISSN: 0890-9369
PB Cold Spring Harbor Laboratory Press
DT Journal
LA English
AB Methylation of histone H3 at Lys 9 is causally linked to formation of
heterochromatin and to long-term transcriptional repression. We report an
unexpected pattern of H3 Lys 9 methylation occurring at a subset of
inducible inflammatory genes. This pattern is characterized by relatively
low constitutive levels of H3 Lys 9 methylation that are erased upon
activation and restored concurrently with post-induction transcriptional
repression. Changes in H3 Lys 9 methylation strongly correlate with RNA
polymerase II recruitment and release. In particular, remethylation
correlates with RNApolII release more strongly than does histone
deacetylation. We propose that, by generating a window of time in which
transcription is permitted, dynamic modulation of H3 Lys 9 methylation
adds an addnl. regulatory level to transcriptional activation of tightly
controlled inducible genes.
OSC.G 84 THERE ARE 84 CAPLUS RECORDS THAT CITE THIS RECORD (84 CITINGS)
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN DUPLICATE 7
AN 2002:576578 BIOSIS
DN PREV200200576578
TI The lymphotoxin-beta receptor induces different patterns of gene
expression via two NF-kappaB pathways.
AU Dejardin, Emmanuel; Droin, Nathalie M.; Delhase, Mireille; Haas, Elvira;
Cao, Yixue; Makris, Constantin; Li, Zhi-Wei; Karin, Michael; Ware, Carl

F.; Green, Douglas R. [Reprint author]
CS Division of Cellular Immunology, La Jolla Institute for Allergy and
Immunology, 10355 Science Center Drive, San Diego, CA, 92121, USA
doug@liai.org
SO Immunity, (October, 2002) Vol. 17, No. 4, pp. 525-535. print.
ISSN: 1074-7613.
DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002
AB The lymphotoxin-beta receptor (LTbetaR) plays critical roles in
inflammation and lymphoid organogenesis through activation of
NF-kappaB.
In addition to activation of the classical NF-kappaB, ligation
of this
receptor induces the processing of the cytosolic NF-kappaB2/p100
precursor
to yield the mature p52 subunit, followed by translocation of
p52 to the
nucleus. This activation of NF-kappaB2 requires NIK and
IKKalpha, while
NEMO/IKKgamma is dispensable for p100 processing.
IKKbeta-dependent
activation of canonical NF-kappaB is required for the expression
but not
processing of p100 and for the expression of proinflammatory
molecules
including VCAM-1, MIP-1beta, and MIP-2 in response to LTbetaR
ligation.
In contrast, IKKalpha controls the induction by LTbetaR ligation
of
chemokines and cytokines involved in lymphoid organogenesis,
including
SLC, BLC, ELC, SDF1, and BAFF.

L13 ANSWER 18 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 8
AN 2002:858817 CAPLUS
DN 137:336538
TI CCL9/MIP-1γ and its receptor CCR1 are the major chemokine
ligand/receptor species expressed by osteoclasts
AU Lean, Jenny M.; Murphy, Chiho; Fuller, Karen; Chambers, Timothy
J.
CS Department of Cellular Pathology, St. George's Hospital Medical
School,
London, SW17 0RE, UK
SO Journal of Cellular Biochemistry (2002), 87(4), 386-393
CODEN: JCEBD5; ISSN: 0730-2312
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Although much has been learned recently of the mechanisms by
which the

differentiation of osteoclasts is induced, less is known of the factors that regulate their migration and localization, and their interactions with other bone cells. In related cell types, chemokines play a major role in these processes. The authors therefore systematically tested the expression of RNA for chemokines and their receptors by osteoclasts. Because bone is the natural substrate for osteoclasts and may influence osteoclast behavior, the authors also tested expression on bone slices. Quant. RT-PCR using real-time anal. with SYBR Green was therefore performed on RNA isolated from bone marrow cells after incubation with macrophage-colony stimulating factor (M-CSF) with/without receptor-activator of NF.kappa.B ligand (RANKL), on plastic or bone. The authors found that RANKL induced expression of CCL9/MIP-1γ to levels comparable to that of tartrate-resistant acid phosphatase (TRAP), a major specialized product of osteoclasts. CCL22/MDC, CXCL13/BLC/BCA-1, and CCL25/TECK were also induced. The dominant chemokine receptor expressed by osteoclasts was CCR1, followed by CCR3 and CX3CR1. Several receptors expressed on macrophages and associated with inflammatory responses, including CCR2 and CCR5, were down-regulated by RANKL. CCL9, which acts through CCR1, stimulated cytoplasmic motility and polarization in osteoclasts, identical to that previously observed in response to CCL3/MIP-1α, which also acts through CCR1 and is chemotactic for osteoclasts. These results identify CCL9 and its receptor CCR1 as the major chemokine and receptor species expressed by osteoclasts, and suggest a crucial role for CCL9 in the regulation of bone resorption.

OSC.G 52 THERE ARE 52 CAPLUS RECORDS THAT CITE THIS RECORD (52 CITINGS)
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 19 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2000:296360 CAPLUS
DN 133:57549

TI Alymphoplasia (aly)-type nuclear factor κB-inducing kinase (NIK) causes defects in secondary lymphoid tissue chemokine receptor signaling and homing of peritoneal cells to the gut-associated lymphatic tissue system

AU Fagarasan, Sidonia; Shinkura, Reiko; Kamata, Tadashi; Nogaki, Fumiaki; Ikuta, Koichi; Tashiro, Kei; Honjo, Tasuku
CS Department of Medical Chemistry Faculty of Medicine, Kyoto University, Kyoto, 606-8501, Japan

SO Journal of Experimental Medicine (2000), 191(9), 1477-1486
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English

AB Alymphoplasia (aly) mice, which carry a point mutation in the nuclear factor κB-inducing kinase (NIK) gene, are characterized by the systemic absence of lymph nodes and Peyer's patches, disorganized splenic and thymic architectures, and immunodeficiency. Another unique feature of aly/aly mice is that their peritoneal cavity contains more B1 cells than normal and aly/+ mice. Transfer expts. of peritoneal lymphocytes from aly/aly mice into recombination activating gene (RAG)-2-/- mice revealed that B and T cells fail to migrate to other lymphoid tissues, particularly to the gut-associated lymphatic tissue system. In vivo homing defects of aly/aly peritoneal cells correlated with reduction of their in vitro chemotactic responses to secondary lymphoid tissue chemokine (SLC) and B lymphocyte chemoattractant (BLC). The migration defect of aly/aly lymphocytes was not due to a lack of expression of chemokines and their receptors, but rather to impaired signal transduction downstream of the receptors for SLC, indicating that NIK is involved in the chemokine signaling pathway known to couple only with G proteins. The results showed that the reduced serum levels of Igs and the absence of class switch to IgA in aly/aly mice are due, at least in part, to a migration defect of lymphocytes to the proper microenvironment where B cells

proliferate and differentiate into Ig-producing cells.
OSC.G 76 THERE ARE 76 CAPLUS RECORDS THAT CITE THIS RECORD (76 CITINGS)
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 20 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2000:885658 CAPLUS
DN 135:45130
TI Mechanism of B1 cell differentiation and migration in GALT
AU Fagarasan, Sidonia; Shinkura, Reiko; Kamata, Tadashi; Nogaki, Fumiaki;
Ikuta, Koichi; Honjo, Tasuku
CS Department of Medical Chemistry, Kyoto University Faculty of Medicine,
Japan
SO Current Topics in Microbiology and Immunology (2000), 252(B1 Lymphocytes
in B Cell Neoplasia), 221-229
CODEN: CTMIA3; ISSN: 0070-217X
PB Springer-Verlag
DT Journal
LA English
AB A study was conducted to investigate the homing capacity of peritoneal
cavity (PEC) cells from aly/aly and aly/+ mice. It was found that PEC
cells from aly/aly mice have a defect in homing to other lymphoid tissues,
and this defect was more severe regarding their migration to the
gut-associated lymphatic tissue system. In vivo migration defect correlated
with in vitro decrease of chemotactic activity of SLC (secondary
lymphoid-tissue chemokine) and BLC (B lymphocyte
chemoattractant) on aly/aly PEC cells. The defective chemotactic response
of aly/aly PEC lymphocytes was not due to the lack of chemokine or their
receptors but to a defect in signaling pathway through the chemokine
receptors. It was observed that the aly mutation of the NF-.
kappa.B-inducing kinase (NIK) gene blocks signaling from
the receptors for SLC, providing the first evidence that NIK is involved
in signal transduction through seven-transmembrane protein receptors.
OSC.G 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 9

AN 1999:810216 CAPLUS
DN 132:106889
TI Distinct activities of p52/NF-.kappa.B required for proper secondary lymphoid organ microarchitecture: functions enhanced by Bcl-3
AU Poljak, Ljiljana; Carlson, Louise; Cunningham, Kirk; Kosco-Vilbois, Marie H.; Siebenlist, Ulrich
CS Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD, 20892, USA
SO Journal of Immunology (1999), 163(12), 6581-6588
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB Mice rendered deficient in p52, a subunit of NF-.kappa .B, or in Bcl-3, an IκB-related regulator that assocs. with p52 homodimers, share defects in the microarchitecture of secondary lymphoid organs. The mutant mice are impaired in formation of B cell follicles and are unable to form proper follicular dendritic cell (FDC) networks upon antigenic challenge. The defects in formation of B cell follicles may be attributed, at least in part, to impaired production of the B lymphocyte chemoattractant (BLC) chemokine, possibly a result of defective FDCs. The p52- and Bcl-3-deficient mice exhibit addnl. defects within the splenic marginal zone, including reduced nos. of metallophilic macrophages, reduced deposition of the laminin-β2 chain and impaired expression of a mucosal addressin marker on sinus-lining cells. Whereas p52-deficient mice are severely defective in all of these aspects, Bcl-3-deficient mice are only partially defective. We determined that FDCs or other non-hemopoietic cells that underlie FDCs are intrinsically impaired in p52-deficient mice. Adoptive transfers of wild-type bone marrow into p52-deficient mice failed to restore FDC networks or follicles. The transfers did restore metallophilic macrophages to the marginal zone, however. Together, the results suggest that p52 carries out functions

essential for a proper splenic microarchitecture in both hemopoietic and
nonhemopoietic cells and that Bcl-3 is important in enhancing these
essential activities of p52.
OSC.G 50 THERE ARE 50 CAPLUS RECORDS THAT CITE THIS RECORD (50 CITINGS)
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2010 ACS on STN
AN 1997:568166 CAPLUS
DN 127:215961
OREF 127:41909a,41912a
TI Gene therapy of endothelial cells with anti-apoptotic proteins for
transplantation and inflammatory conditions
IN Bach, Fritz H.; Ferran, Christiane
PA Novartis A.-G., Switz.; New England Deaconess Hospital Corporation; Bach,
Fritz H.; Ferran, Christiane
SO PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9730083	A1	19970821	WO 1997-EP676	19970213
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2245503	A1	19970821	CA 1997-2245503	19970213
AU 9718730	A	19970902	AU 1997-18730	19970213
EP 886650	A1	19981230	EP 1997-905019	19970213
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

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             IE, SI, FI, RO
     JP 2000510326        T     20000815    JP 1997-528990
19970213
PRAI US 1996-601515       A     19960214
     US 1996-634995       A     19960419
     WO 1997-EP676        W     19970213
AB   A method of genetically modifying mammalian, especially
endothelial cells to
     render them less susceptible to an inflammatory or other immunol.
     activation stimulus is described, which comprises inserting in
that cell
     or a progenitor thereof DNA encoding an anti-apoptotic
polypeptide capable
     of inhibiting NF-.kappa.B and expressing the
     protein, whereby NF-.kappa.B in the cell is
     substantially inhibited in the presence of a cellular activating
stimulus.
     Suitable polypeptides are selected from those having activity of
a
     mammalian A20, BCL-2, BCL-XL (MCL-1) or A1 protein, including
homologs and
     truncated forms of the native proteins.  The BCL-2, BCL-XL or A1
active
     polypeptides can also be employed as homodimers or as
heterodimers with
     another anti-apoptotic polypeptide of the BCL family.  The
method, which
     can be carried out in vivo or ex vivo or in vitro, is
particularly useful
     in connection with allogeneic or, especially, xenogeneic
transplantation, as
     well as to treat systemic or local inflammatory conditions.
Transgenic or
     somatic recombinant non-human mammals can be prepared expressing
such a
     polypeptide on a regulable basis by the endothelial cells
thereof, and
     tissues or organs comprising such cells can be obtained for
grafting into
     a mammalian recipient.  An example illustrating the invention is
     transformation of endothelial cells to recombinantly express
BCL-2 and
     BCL-XL.  Transcription factor NF-.kappa.B
     was inhibited in these cells as demonstrated using reporter
genes.
OSC.G   7     THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7
CITINGS)
RE.CNT  6     THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> FIL STNGUIDE
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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.42     175.03

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
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=> d his

     (FILE 'HOME' ENTERED AT 12:06:56 ON 20 APR 2010)

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:15:37 ON 20 APR 2010
L1             3 S (BLC OR ELC) (3A) PROMOTER
L2             3 DUP REM L1 (0 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:22:26 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:23:18 ON 20 APR 2010
L3             1 S NF KAPP B
L4        113335 S NF KAPPA B
L5            36 S L4 AND (BLC OR ELC)
L6             5 S L5 AND PROMOTER
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L7             4 DUP REM L6 (1 DUPLICATE REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:28:53 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:29:56 ON 20 APR 2010
L8          2987 S CCL21 OR CXCL13
L9            11 S L8 (3A) PROMOTER
L10            5 DUP REM L9 (6 DUPLICATES REMOVED)
L11          110 S L4 AND L8
L12            5 S L11 AND PROMOTER
L13           22 DUP REM L5 (14 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 12:35:33 ON 20 APR 2010

     FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:38:56 ON 20 APR 2010

=> s l11 and py<=2004
L14          21 L11 AND PY<=2004

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15           11 DUP REM L14 (10 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 11 ANSWERS - CONTINUE? Y/(N):y

L15  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2010 ACS on STN
AN   2007:647385  CAPLUS
DN   147:87651
TI   Gene expression profiles to identify effectors of innate
immunity for the
     treatment of inflammation or sepsis
IN   Hancock, Robert E.W.; Finlay, B. Brett; Gough Scott, Monisha;
Bowdish,
     Dawn; Rosenberger, Carrie Melissa; Steven Powers, Jon-Paul; Yu,
Jie;
     Mookherjee, Neeloffer
PA   University of British Columbia, Can.
SO   U.S. Pat. Appl. Publ., 213 pp., Cont.-in-part of U.S. Ser. No.
241,882.
     CODEN: USXXCO
DT   Patent
LA   English
FAN.CNT 4
     PATENT NO.          KIND   DATE        APPLICATION NO.
DATE
     ---------------     ----   --------    --------------------
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PI   US 20070134261       A1    20070614    US 2006-400411
20060407
     US 20040001803       A1    20040101    US 2002-308905
20021202 <--
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US 7507787 B2 20090324
CN 101215601 A 20080709 CN 2007-10168028
20021202
NZ 563261 A 20080829 NZ 2002-563261
20021202
US 20040180038 A1 20040916 US 2003-661471
20030912 <--
US 7687454 B2 20100330
US 20070190533 A1 20070816 US 2005-241882
20050929
AU 2007201885 A1 20070517 AU 2007-201885
20070427
PRAI US 2001-336632P P 20011203
US 2002-308905 A2 20021202
US 2003-661471 A2 20030912
US 2005-241882 A2 20050929
AU 2002-365675 A3 20021202
CN 2002-827327 A3 20021202
NZ 2002-533721 A3 20021202
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB This invention is based on the discovery that based on patterns
of
polynucleotide expression regulated by endotoxic
lipopolysaccharide,
lipoteichoic acid, CpG DNA, or other cellular components (e.g.,
microbes),
and affected by cationic peptides, one can screen for novel
compds. that
block or reduce sepsis and/or inflammation in a subject. The
method
includes contacting cells with lipopolysaccharide, lipoteichoic
acid, CpG
DNA, and/or intact microbes or microbial components in the
presence or
absence of a peptide; detecting a pattern of polynucleotide
expression for
the cells in the presence and absence of the peptide, wherein
the pattern
in the presence of the peptide represents inhibition of an
inflammatory or
septic response. A method of identifying a polynucleotide or
pattern of
polynucleotides regulated by one or more sepsis or inflammatory
inducing
agents and inhibited by a peptide is described. In another
aspect, the
invention provides methods and compds. for enhancing innate
immunity in a
subject. Based on the use of cationic peptides as a tools, one
can
identify selective enhancers of innate immunity that do not
trigger the

sepsis reaction and that can block/dampen inflammatory and/or septic
responses. A method of selectively suppressing sepsis is provided, while
maintaining expression of an anti-inflammatory gene. Cationic peptides,
such as human cathelicidin LL-37 or KSRIVPAIPVSLL and related peptides,
are provided for protection against bacterial infection by enhancing
immune response via down-regulation of pro-inflammatory genes and
up-regulation of anti-inflammatory genes.
OSC.G 0 THERE ARE 0 CAPLUS RECORDS THAT CITE THIS RECORD (0 CITINGS)

L15 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 1
AN 2004:927620 CAPLUS
DN 142:5222
TI IκB Kinase Complex α Kinase Activity Controls Chemokine and
High Endothelial Venule Gene Expression in Lymph Nodes and
Nasal-Associated Lymphoid Tissue
AU Drayton, Danielle L.; Bonizzi, Giuseppina; Ying, Xiaoyan; Liao, Shan;
Karin, Michael; Ruddle, Nancy H.
CS Department of Epidemiology and Public Health, Section of Immunobiology,
Yale University School of Medicine, New Haven, CT, 06520, USA
SO Journal of Immunology (2004), 173(10), 6161-6168
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The lymphotoxin (LT) β receptor plays a critical role in secondary
lymphoid organogenesis and the classical and alternative NF-.
kappa.B pathways have been implicated in this process.
IKKα is a key mol. for the activation of the alternative NF
-.kappa.B pathway. However, its precise role and
target genes in secondary lymphoid organogenesis remain unknown,
particularly with regard to high endothelial venules (HEV). In this
study, we show that IKKαAA mutant mice, who lack inducible kinase
activity, have hypocellular lymph nodes (LN) and nasal-associated lymphoid
(NALT) tissue characterized by marked defects in microarchitecture and
HEV. In addition, IKKαAA LNs showed reduced lymphoid chemokine CCL19,
CCL21, and CXCL13 expression. IKKαAA LN- and
NALT-HEV were abnormal in appearance with reduced expression of peripheral

node addressin (PNAd) explained by a severe reduction in the HEV-associated
proteins, glycosylation-dependent cell adhesion mol. 1 (GlyCAM-1), and
high endothelial cell sulfotransferase, a PNAd-generating enzyme that is a
target of LTαβ. In this study, anal. of LTβ-/- mice
identifies GlyCAM-1 as another LTβ-dependent gene. In contrast,
TNFRI-/- mice, which lose classical NF-κ
B pathway activity but retain alternative NF-.
kappa.B pathway activity, showed relatively normal
GlyCAM-1 and HEC-6ST expression in LN-HEV. In addition, in this
communication, it is demonstrated that LTβR is prominently expressed
on LN- and NALT-HEV. Thus, these data reveal a critical role for IKKα
in LN and NALT development, identify GlyCAM-1 and high endothelial cell
sulfotransferase as new IKKα-dependent target genes, and suggest
that LTβR signaling on HEV can regulate HEV-specific gene expression.
OSC.G 26 THERE ARE 26 CAPLUS RECORDS THAT CITE THIS RECORD (26 CITINGS)
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 2
AN 2004:230483 CAPLUS
DN 140:269393
TI Impaired lymphoid chemokine-mediated migration due to a block on the
chemokine receptor switch in human cytomegalovirus-infected dendritic
cells
AU Moutaftsi, Magdalena; Brennan, Paul; Spector, Stephen A.; Tabi, Zsuzsanna
CS Section of Infection and Immunity, University of Wales College of Medicine, Cardiff, CF14 2TL, UK
SO Journal of Virology (2004), 78(6), 3046-3054
CODEN: JOVIAM; ISSN: 0022-538X
PB American Society for Microbiology
DT Journal
LA English
AB Dendritic cell (DC) migration from the site of infection to the site of
T-cell priming is a crucial event in the generation of antiviral T-cell
responses. Here we present to our knowledge the first functional evidence
that human cytomegalovirus (HCMV) blocks the migration of infected
monocyte-derived DCs toward lymphoid chemokines CCL19 and CCL21.

DC migration is blocked by viral impairment of the chemokine receptor
switch at the level of the expression of CCR7 mols. The inhibition occurs
with immediate-early-early kinetics, and viral interference with NF-.kappa.B signaling is likely to be at least
partially responsible for the lack of CCR7 expression. DCs which migrate
from the infected cultures are HCMV antigen neg., and consequently they do
not stimulate HCMV-specific CD8+ T cells, while CD4+-T-cell activation is
not impaired. Although CD8+ T cells can also be activated by alternative
antigen presentation mechanisms, the spatial segregation of naive T cells
and infected DCs seems a potent mechanism of delaying the generation of
primary CD8+-T-cell responses and aiding early viral spread.
OSC.G 20 THERE ARE 20 CAPLUS RECORDS THAT CITE THIS RECORD (20 CITINGS)
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 11 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3
AN 2004:405713 BIOSIS
DN PREV200400408862
TI A stroma-derived defect in NF-kappaB2-/- mice causes impaired lymph node
development and lymphocyte recruitment.
AU Carragher, Damian; Johal, Ramneek; Button, Adele; White, Andrea;
Eliopoulos, Aristides; Jenkinson, Eric; Anderson, Graham; Caamano, Jorge
[Reprint Author]
CS Sch MedMRCCtr Immune Regulat, Univ Birmingham, Birmingham, W Midlands, B15
2TT, UK
J.Caamano@bham.ac.uk
SO Journal of Immunology, (August 15 2004) Vol. 173, No. 4, pp. 2271-2279. print.
ISSN: 0022-1767 (ISSN print).
DT Article
LA English
ED Entered STN: 20 Oct 2004
Last Updated on STN: 20 Oct 2004
AB The NF-kappaB family of transcription factors is vital to all aspects of
immune function and regulation in both the hemopoietic and stromal
compartments of immune environments. Recent studies of mouse models

deficient for specific members of the NF-kappaB family have revealed critical roles for these proteins in the process of secondary lymphoid tissue organogenesis. In this study, we investigate the role of NF-kappaB family member NF-kappaB2 in lymph node development and lymphocyte recruitment. Inguinal lymph nodes in nfkappab2-/- mice are reduced in size and cellularity, most notably in the B cell compartment. Using in vitro and in vivo lymph node grafting assays, we show that the defect resides in the stromal compartment. Further examination of the nfkappab2-/- inguinal lymph nodes revealed that expression of peripheral node addressin components CD34 and glycosylation-dependent cell adhesion molecule-1 along with the high endothelial venule-restricted sulfoltransferase HEC-GlcNAc6ST was markedly reduced. Furthermore, expression of the lymphocyte homing chemokines CCL19, CCL21, and CXCL13 was down-regulated. These data highlight the role of NF-kappaB2 in inguinal lymph node organogenesis and recruitment of lymphocytes to these organs due to its role in up-regulation of essential cell adhesion molecules and chemokines, while suggesting a potential role for NF-kappaB2 in organization of lymph node endothelium.

L15 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:373928 CAPLUS
DN 141:86767
TI Transcriptional profiling reveals suppressed erythropoiesis, up-regulated glycolysis, and interferon-associated responses in murine malaria
AU Sexton, Adrienne C.; Good, Robert T.; Hansen, Diana S.; D'Ombrain, Marthe C.; Buckingham, Lynn; Simpson, Ken; Schofield, Louis
CS The Walter and Eliza Hall Institute of Medical Research, Parkville, Australia
SO Journal of Infectious Diseases (2004), 189(7), 1245-1256
CODEN: JIDIAQ; ISSN: 0022-1899
PB University of Chicago Press
DT Journal
LA English
AB The primary pathophysiol. events contributing to fatal malaria are the cerebral syndrome, anemia, and lactic acidosis. The mol. basis of each

event was unclear. In the present study, microarray anal. of murine
transcriptional responses during the development of severe disease
revealed temporal, organ-specific, and pathway-specific patterns. More
than 400 genes in the brain and 600 genes in the spleen displayed
transcriptional changes. Dominant patterns revealed strongly suppressed
erythropoiesis, starting early during infection, and highly up-regulated
transcription of genes that control host glycolysis, including lactate
dehydrogenase. The latter presents a mechanism that may contribute to
metabolic acidosis. No evidence for hypoxia-mediated regulation of these
events was observed Interferon-regulated gene transcripts dominated the
inflammatory response to cytokines. These results demonstrate previously
unknown transcriptional changes in the host that may underlie the
development of malarial syndromes, such as anemia and metabolic
dysregulation, and increase the utility of murine models in investigation
of basic malarial pathogenesis.
OSC.G 42 THERE ARE 42 CAPLUS RECORDS THAT CITE THIS RECORD (42 CITINGS)
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:308816 CAPLUS
DN 140:301754
TI Injury-induced NF-.kappa.B activation in the
hippocampus: implications for neuronal survival
AU Kassed, Cheryl A.; Butler, Tanya L.; Patton, Geoffrey W.; De Mesquita,
Dirson D.; Navidomskis, Matthew T.; Memet, Sylvie; Israeel, Alain;
Pennypacker, Keith R.
CS Dep. of Pharmacol. and Therapeutics, Univ. of South Florida, Tampa, FL,
33612, USA
SO FASEB Journal (2004), 18(6), 723-724, 10.1096/fj.03-0773fje
CODEN: FAJOEC; ISSN: 0892-6638
PB Federation of American Societies for Experimental Biology
DT Journal
LA English
AB Nuclear factor (NF)-.kappa.B p50 protein is
involved in promoting survival in hippocampal neurons after trimethyltin

(TMT)-injury. In the current study, hippocampal NF-. kappa.B activity was examined and quantitated from transgenic κB-lacZ reporter mice after chemical-induced injury. NF-.kappa.B activity was localized primarily to hippocampal neurons and significantly elevated over that in saline-treated mice between 4 and 21 days after TMT injection. Seven days after TMT injection, a time-point of elevated NF-.kappa .B activity, gene expression in the hippocampus was studied by microarray anal. through comparison of expression profiles between treated nontransgenic and p50-null mice with their saline-injected controls. Seventeen genes increased in nontransgenic TMT-treated mice relative to saline-treated as well as showing no increase in p50-null mice, indicating a role for p50 in their regulation. One of these genes, the Na+,K+-ATPase-γ subunit, was detected in brain for the first time. Several of the genes modulated by NF-.kappa.B are potentially related to neuroplasticity, providing addnl. evidence that this transcription factor is a neuroprotective signal in the hippocampus.
OSC.G 24 THERE ARE 24 CAPLUS RECORDS THAT CITE THIS RECORD (24 CITINGS)
RE.CNT 120 THERE ARE 120 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 7 OF 11 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 4
AN 2004:389553 BIOSIS
DN PREV200400388523
TI Chemokine receptor CCR7 induces intracellular signaling that inhibits apoptosis of mature dendritic cells.
AU Sanchez-Sanchez, Noelia; Riol-Blanco, Lorena; de la Rosa, Gonzalo; Puig-Kroger, Amaya; Garcia-Bordas, Julio; Martin, Daniel; Longo, Natividad; Cuadrado, Antonio; Cabanas, Carlos; Corbi, Angel L.; Sanchez-Mateos, Paloma; Rodriguez-Fernandez, Jose Luis [Reprint Author]
CS Ctr Invest Biol, CSIC, C Ramiro de Maeztu 9, Madrid, 28040, Spain rodrifer@cib.csic.es
SO Blood, (August 1 2004) Vol. 104, No. 3, pp. 619-625. print. CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 6 Oct 2004
Last Updated on STN: 6 Oct 2004

AB Acquisition of CCR7 expression is an important phenotype change during dendritic. cell (DC) maturation that endows these cells with the capability to migrate to lymph nodes. We have analyzed the possible role of CCR7 on the regulation of the survival of DCs. Stimulation with CCR7 ligands CCL19 and CCL21 inhibits apoptotic hallmarks of serum-deprived DCs, including membrane phosphatidylserine exposure, loss of mitochondria membrane potential, increased membrane blebs, and nuclear changes. Both chemokines induced a rapid activation of phosphatidylinositol 3'-kinase/Akt1 (PI3K/Akt1), with a prolonged and persistent activation of Akt1. Interference with PI3K, Gi, or G protein betagamma subunits abrogated the effects of the chemokines on Akt1 activation and on survival. In contrast, inhibition of extracellular signal-related kinase 1/2 (Erk1/2), p38, or c-Jun N-terminal kinase (JNK) was ineffective. Nuclear factor-kappaB (NFkappaB) was involved in the antiapoptotic effects of chemokines because inhibition of NFkappaB blunted the effects of CCL19 and CCL21 on survival. Furthermore, chemokines induced down-regulation of the NFkappaB inhibitor IkappaB, an increase of NFkappaB DNA-binding capability, and translocation of the NFkappaB subunit p65 to the nucleus. In summary, in addition to its well-established role in chemotaxis, we show that CCR7 also induces antiapoptotic signaling in mature DCs.

L15 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 5
AN 2003:911958 CAPLUS
DN 140:40609
TI Differential regulation of CCL21 in lymphoid/nonlymphoid tissues for effectively attracting T cells to peripheral tissues
AU Lo, James C.; Chin, Robert K.; Lee, Youjin; Kang, Hyung-sik; Wang, Yang; Weinstock, Joel V.; Banks, Theresa; Ware, Carl F.; Franzoso, Guido; Fu, Yang-xin
CS Committee on Immunology, University of Chicago, Chicago, IL, USA
SO Journal of Clinical Investigation (2003), 112(10), 1495-1505
CODEN: JCINAO; ISSN: 0021-9738
PB American Society for Clinical Investigation

DT Journal
LA English
AB CC chemokine ligand 21 (CCL21)/secondary lymphoid chemokine (SLC), a ligand for CC chemokine receptor 7 (CCR7), has been demonstrated to play a vital role in the homing and localization of immune cells to lymphoid tissues, but its role in nonlymphoid tissues largely remains undefined. Here, we provide evidence that CCL21 in lymphoid and nonlymphoid tissues is differentially regulated by lymphotoxin-dependent (LT-dependent) and -independent mechanisms, resp. This differential regulation is due to the selective regulation of the CCL21 -Ser/CCL21a but not the CCL21-Leu/CCL21b gene by the LT and noncanonical NF-.kappa.B pathways. This alternate pathway, not dependent on LT or lymphocytes, leading to constitutive expression of CCL21 in nonlymphoid tissues, is critical for the initial recruitment of T lymphocytes to peripheral effector sites. CCL21 expression is subsequently further enhanced in a LT-dependent fashion following airway challenge, potentially facilitating a pos. feedback loop to attract addnl. CCR7+ effector cells. These findings establish an essential role for CCL21 in the recruitment of effector T cells to peripheral tissues and suggest that LT-dependent and -independent regulation of CCL21 plays a role in balancing the central and peripheral immune responses between lymphoid and nonlymphoid tissues.
OSC.G 38 THERE ARE 38 CAPLUS RECORDS THAT CITE THIS RECORD (38 CITINGS)
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2003:854656 CAPLUS
DN 140:58113
TI The chemokine CCL21 modulates lymphocyte recruitment and fibrosis in chronic hepatitis C
AU Bonacchi, Andrea; Petrai, Ilaria; De Franco, Raffaella M. S.; Lazzeri, Elena; Annunziato, Francesco; Efsen, Eva; Cosmi, Lorenzo; Romagnani, Paola; Milani, Stefano; Failli, Paola; Batignani, Giacomo; Liotta, Francesco; Laffi, Giacomo; Pinzani, Massimo; Gentilini, Paolo; Marra, Fabio

CS Dipartimento di Medicina Interna, University of Florence, Florence, Italy
SO Gastroenterology (2003), 125(4), 1060-1076
CODEN: GASTAB; ISSN: 0016-5085
PB W. B. Saunders Co.
DT Journal
LA English
AB The chemokines CCL19 and CCL21 bind CCR7, which is involved in the organization of secondary lymphoid tissue and is expressed during chronic tissue inflammation. The authors investigated the expression of CCL21 and CCR7 in chronic hepatitis C. The effects of CCL21 on hepatic stellate cells (HSCs) were also studied. Expression of CCL21 was assessed by in situ hybridization and immunohistochem. CCR7 on T cells was analyzed by flow cytometry. Cultured human HSCs were studied in their activated phenotype. In patients with chronic hepatitis C, expression of CCL21 and CCR7 was up-regulated. CCL21 was detected in the portal tracts and around inflammatory lymphoid follicles, in proximity to T lymphocytes and dendritic cells, which contributed to expression of this chemokine. Expression of CCR7 was also increased in patients with primary biliary cirrhosis. Intrahepatic CD8+ T lymphocytes isolated from patients with chronic hepatitis C had a higher percentage of positivity for CCR7 than those from healthy controls, and the expression of CCR7 was associated with that of CXCR3. Cultured HSCs expressed functional CCR7, the activation of which stimulated cell migration and accelerated wound healing in an in vitro model. Exposure of HSCs to CCL21 triggered several signaling pathways, including extracellular signal-regulated kinase, Akt, and nuclear factor κB, resulting in induction of proinflammatory genes. Thus, expression of CCL21 during chronic hepatitis C is implicated in the recruitment of T lymphocytes and the organization of inflammatory lymphoid tissue and may promote fibrogenesis in the inflamed areas via activation of CCR7 on HSCs.
OSC.G 41 THERE ARE 41 CAPLUS RECORDS THAT CITE THIS RECORD (41 CITINGS)
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 10 OF 11 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN DUPLICATE 6
AN 2002:614600 BIOSIS
DN PREV200200614600
TI Long-lived immature dendritic cells mediated by TRANCE-RANK interaction.
AU Cremer, Isabelle; Dieu-Nosjean, Marie-Caroline; Marechal, Sylvie; Dezutter-Dambuyant, Colette; Goddard, Sarah; Adams, David; Winter, Nathalie; Menetrier-Caux, Christine; Sautes-Fridman, Catherine; Fridman, Wolf H.; Mueller, Chris G. F. [Reprint author]
CS Centre de Recherches Biomedicales des Cordeliers, INSERM U255, 15 Rue de l'Ecole de Medecine, Paris Cedex 6, 75270, France chmuller@infobiogen.fr
SO Blood, (November 15, 2002) Vol. 100, No. 10, pp. 3646-3655. print.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 4 Dec 2002
Last Updated on STN: 4 Dec 2002
AB Immature dendritic cells (DCs) reside in interstitial tissues (int-DC) or in the epidermis, where they capture antigen and, thereafter, mature and migrate to draining lymph nodes (LNs), where they present processed antigen to T cells. We have identified int-DCs that express both TRANCE (tumor necrosis factor-related activation-induced cytokine) and RANK (receptor activator of NF-kappaB) and have generated these cells from CD34+ human progenitor cells using macrophage colony-stimulating factor (M-CSF). These CD34+-derived int-DCs, which are related to macrophages, are long-lived, but addition of soluble RANK leads to significant reduction of cell viability and Bcl-2 expression. This suggests that constitutive TRANCE-RANK interaction is responsible for CD34+-derived int-DC longevity. Conversely, CD1a+ DCs express only RANK and are short-lived. However, they can be rescued from cell death either by recombinant soluble TRANCE or by CD34+-derived int-DCs. CD34+-derived int-DCs mature in response to lipopolysaccharide (LPS) plus CD40 ligand (L) and become capable of CCL21/CCL19-mediated chemotaxis and

naive T-cell activation. Upon maturation, they lose TRANCE, making them, like CD1a+ DCs, dependent on exogenous TRANCE for survival. These findings provide evidence that TRANCE and RANK play important roles in the homeostasis of DCs.

L15 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 7
AN 2002:858817 CAPLUS
DN 137:336538
TI CCL9/MIP-1γ and its receptor CCR1 are the major chemokine ligand/receptor species expressed by osteoclasts
AU Lean, Jenny M.; Murphy, Chiho; Fuller, Karen; Chambers, Timothy J.
CS Department of Cellular Pathology, St. George's Hospital Medical School, London, SW17 0RE, UK
SO Journal of Cellular Biochemistry (2002), 87(4), 386-393
CODEN: JCEBD5; ISSN: 0730-2312
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Although much has been learned recently of the mechanisms by which the differentiation of osteoclasts is induced, less is known of the factors that regulate their migration and localization, and their interactions with other bone cells. In related cell types, chemokines play a major role in these processes. The authors therefore systematically tested the expression of RNA for chemokines and their receptors by osteoclasts. Because bone is the natural substrate for osteoclasts and may influence osteoclast behavior, the authors also tested expression on bone slices. Quant. RT-PCR using real-time anal. with SYBR Green was therefore performed on RNA isolated from bone marrow cells after incubation with macrophage-colony stimulating factor (M-CSF) with/without receptor-activator of NF.kappa.B ligand (RANKL), on plastic or bone. The authors found that RANKL induced expression of CCL9/MIP-1γ to levels comparable to that of tartrate-resistant acid phosphatase (TRAP), a major specialized product of osteoclasts. CCL22/MDC, CXCL13/BLC/BCA-1, and CCL25/TECK were also induced. The dominant chemokine receptor expressed by osteoclasts

was CCR1, followed by CCR3 and CX3CR1. Several receptors expressed on
macrophages and associated with inflammatory responses, including CCR2 and
CCR5, were down-regulated by RANKL. CCL9, which acts through CCR1,
stimulated cytoplasmic motility and polarization in osteoclasts, identical
to that previously observed in response to CCL3/MIP-1α, which also acts
through CCR1 and is chemotactic for osteoclasts. These results identify
CCL9 and its receptor CCR1 as the major chemokine and receptor species
expressed by osteoclasts, and suggest a crucial role for CCL9 in the
regulation of bone resorption.
OSC.G 52 THERE ARE 52 CAPLUS RECORDS THAT CITE THIS RECORD (52 CITINGS)
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 16, 2010 (20100416/UP).

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.28	215.11

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	

-21.25

STN INTERNATIONAL LOGOFF AT 12:44:30 ON 20 APR 2010